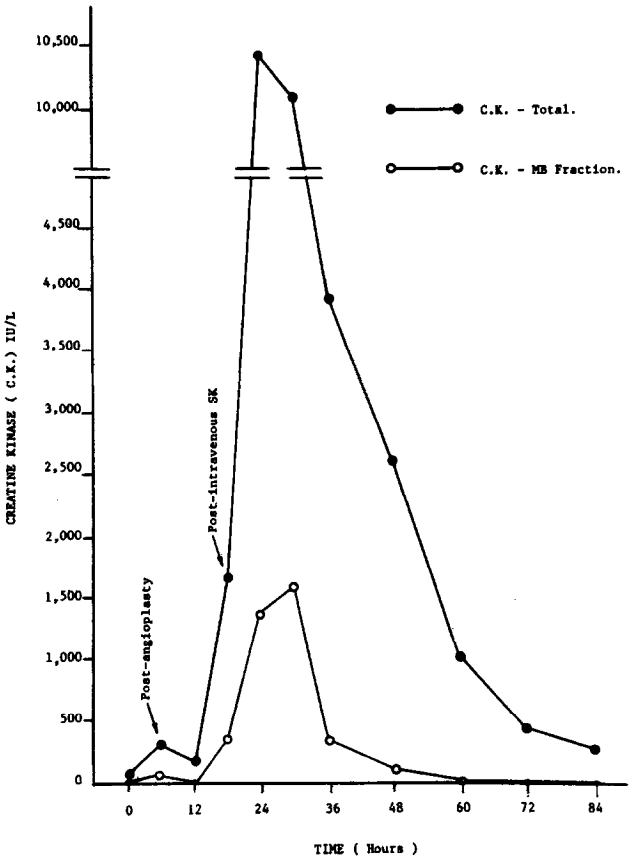

Fig. 2. Creatine kinase *(C.K.)* measurements demonstrating the rapid increase shortly after the onset of intravenous streptokinase *(SK)* administration. *Closed circles* represent total C.K. and *open circles* represent C.K.-MB fraction.

emergency coronary artery bypass graft surgery, but only after reversing the fibrinolytic effect with fresh frozen plasma.[7] Intracoronary streptokinase has been given to patients who were on the IABCP with no significant bleeding complications, but the dose used was smaller than that considered necessary to open a thrombosed coronary using intravenous administration of streptokinase.[8] It was felt that our patient was clinically too unstable to tolerate the delay associated with catheterization. Since it was likely that the reocclusion was due to thrombosis, we felt that the risk of intravenous streptokinase was justified and that the potential benefit of intravenous streptokinase outweighed the risk of bleeding. We proceeded with intravenous streptokinase infusion. The patient tolerated the procedure without bleeding or other complications in the presence of the IABCP. His prompt improvement and the apparent rapid washout of creatine phosphokinase after administration of streptokinase support (but do not prove)[2] that the reocclusion was most likely secondary to new clot formation and that the intravenous enzyme reestablished patency. Because the entire episode was probably initiated by thrombotic occlusion, the patient was continued on anticoagulation with heparin and subsequently with warfarin sodium and dipyridamole. Although we encountered no serious complications from the use of intravenous streptokinase in the presence of IABCP in our patient, we would suggest caution in the use of this therapeutic combination. At 4 months post angioplasty and streptokinase, he continues to do well, with no angina but with mild cardiac failure.

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Long-term follow-up of coronary artery occlusion secondary to blunt chest trauma

Joel K. Kahn, M.D., and Andrew J. Buda, M.D.
Ann Arbor, Mich.

Transmural myocardial infarction following nonpenetrating chest trauma has been described,[1,2] but demonstration of simultaneous coronary occlusion has been infrequently reported.[3-5] In this report, we describe the clinical, scintigraphic, echocardiographic, angiographic, and long term follow-up findings in a 30-year-old man who suffered an

From the Department of Internal Medicine, Division of Cardiology, University of Michigan Medical School.

Reprint requests: Andrew J. Buda, M.D., Cardiology Division, 3910 Alfred Taubman Care Center, University of Michigan Medical Center, 1405 E. Ann St., Ann Arbor, MI 48109-0336.

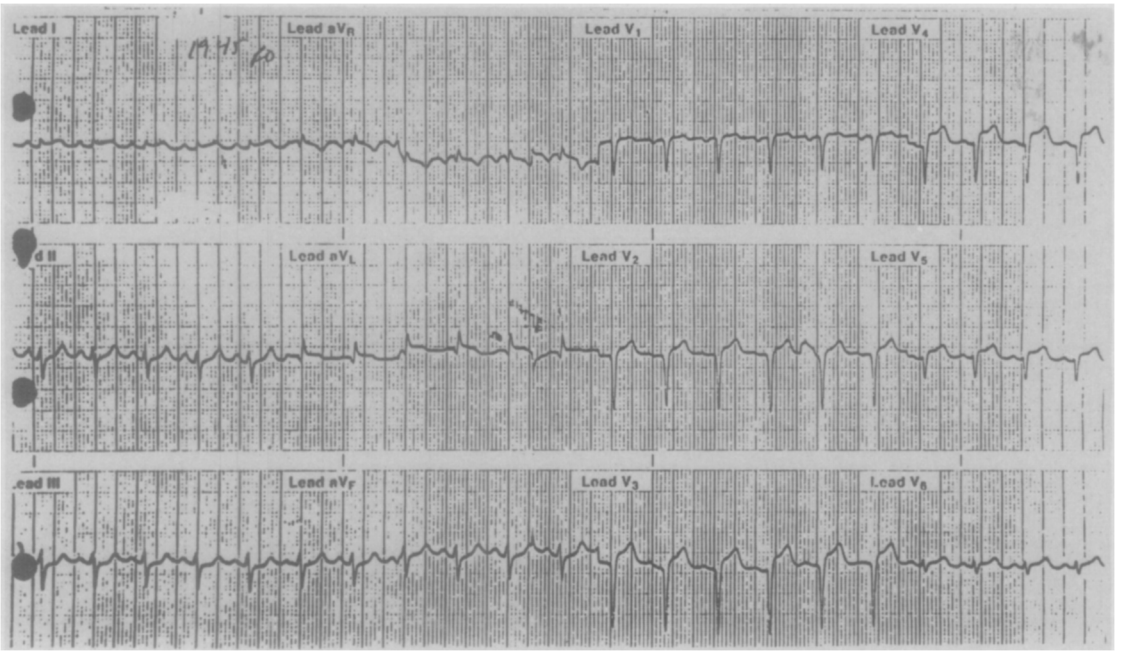

Fig. 1. Admission electrocardiogram revealing sinus tachycardia, Q waves in leads 1, aV_L, and V_1 through V_4, and ST segment elevation in leads 1, aV_L and V_1 through V_6.

extensive anterolateral myocardial infarction following a motor vehicle accident.

On March 30, 1983, a healthy 30-year-old man sustained blunt chest trauma in a high speed motor vehicle accident. Following stabilization at a local emergency room, he was transferred to the University of Michigan Medical Center for further care. On arrival, he had a blood pressure of 108/80 mm Hg in both arms, a heart rate of 120 bpm, and respirations of 24 per minute. Examination revealed subcutaneous emphysema, marked tenderness over the left anterior chest, and equal pulses. A chest roentgenogram revealed a right apical pulmonary contusion and a widened mediastinum. An ECG revealed sinus tachycardia with Q waves in leads I, aV_L, and V_1 to V_4, and ST segment elevation in leads I, aV_L, and V_1 through V_6 (Fig. 1). An emergent thoracic aortogram did not reveal a dissection. The patient was admitted to the Intensive Care Unit and a pulmonary artery flotation catheter revealed a right atrial pressure of 16 mm Hg, a mean pulmonary artery pressure of 26 mm Hg, a pulmonary artery occlusion pressure of 23 mm Hg, and a cardiac output of 5.9 L/min. The creatine phosphokinase curve peaked at 10,000 IU/L with a positive MB isoenzyme fraction determined by gel electrophoresis. Rest thallium myocardial perfusion scintigraphy demonstrated markedly decreased tracer uptake in the septum and apical areas, with lesser perfusion defects in the anterior and apicoinferior segments, without change on delayed images (Fig. 2). Gated equilibrium blood pool radionuclide ventriculography performed the day following admission revealed an ejection fraction of 16% with an enlarged left ventricle. Two-dimensional and M-mode echocardiography showed a left ventricular end-diastolic diameter at the upper limits of normal (57 mm), hypokinesis of the septal, anterior and anterolateral segments, and no pericardial effusion.

The patient's dyspnea resolved on the second hospital day. He remained hemodynamically stable, but a third and fourth heart sound developed. On the eleventh hospital day, cardiac catheterization revealed a pulmonary artery pressure of 24/13 mm Hg, a pulmonary artery occlusion pressure of 14 mm Hg, a left ventricular end-diastolic pressure of 30 mm Hg before contrast, and a cardiac index of 4.2 L/min/m^2. Left ventriculography showed a dilated left ventricle with akinesis of the anterolateral and apical segments and an ejection fraction of 15%. There was no valvular regurgitation. Coronary arteriography revealed normal right, circumflex, and left main coronary circulations. The left anterior descending artery had an abrupt 90% short stenosis in the proximal segment, suggesting a tear to the vessel wall (Fig. 3), with a normal distal vessel. On the fifteenth hospital day, the day of discharge, a resting thallium myocardial perfusion scintigram was unchanged from admission. When seen 1 month after discharge, the patient had no complaints of shortness of breath or chest pain consistent with myocardial ischemia. He was bicycling ½ mile daily without difficulty. When seen 24 months after discharge, the patient was experiencing occasional dyspnea at rest but continued to exercise regularly. A fourth heart sound was present. A Bruce protocol exercise treadmill test revealed an exercise duration of 18 minutes without ECG changes. Radionuclide ventriculography revealed a rest ejection fraction of 21%, rising to 23% with a workload of 125 W. Echocardiography demonstrated left ventricular enlargement, with an end-diastolic diameter of 70 mm.

Myocardial contusion is the most frequent injury sus-

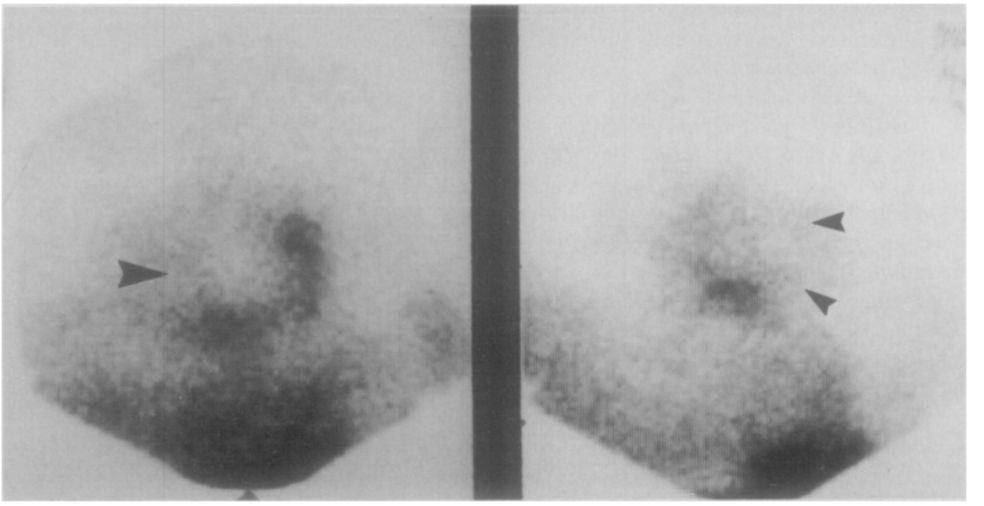

Fig. 2. Rest thallium myocardial perfusion scintigram in the anterior *(right)* and 40-degree left anterior oblique *(left)* projections. There is marked decreased tracer uptake in the septal and apical segments *(arrow, left panel)*, with less severe perfusion defects in the anterior and apicoinferior portions of the left ventricle *(arrows, right panel)*.

tained following nonpenetrating chest trauma.[1] Electrocardiography has been used to diagnose myocardial contusion with high sensitivity,[6] although the ECG obtained soon after the injury often reveals nonspecific changes.[1] Determination of creatine kinase MB isoenzyme has been used to identify the presence of myocardial injury, and may enhance the specificity of ECG abnormalities observed.[7] Infarct-avid myocardial scanning, radionuclide ventriculography, and two-dimensional echocardiography have also been used to identify myocardial dysfunction or necrosis secondary to blunt cardiac trauma; however, a recent prospective study[8] found that all noninvasive methods studies were of poor diagnostic accuracy. Despite a high diagnostic accuracy in the setting of acute myocardial infarction secondary to atherosclerotic coronary artery disease, the immediate utilization of thallium perfusion scintigraphy to establish the presence and extent of myocardial ischemia and necrosis has not previously been emphasized in the setting of suspected traumatic cardiac injury

Transmural myocardial infarction due to coronary artery occlusion following blunt chest trauma has been considered a rare event. Parmley et al.[1] identified no cases of coronary artery occlusion among 546 autopsy cases of blunt chest trauma. A recent review of the literature[3] identified only six cases of angiographically confirmed coronary artery obstruction following nonpenetrating chest trauma. Three additional cases have recently been reported.[4,5] Complications within the first month of traumatic myocardial infarction were frequent and included complex arrhythmias,[3] the development of a ventricular septal defect[4] and ventricular aneurysms,[3,4] and sudden death.[3] The follow-up and long-term course of these patients has included full recovery,[3,5] sudden death,[3] crippling angina,[3] and severe congestive failure.[3] Coronary

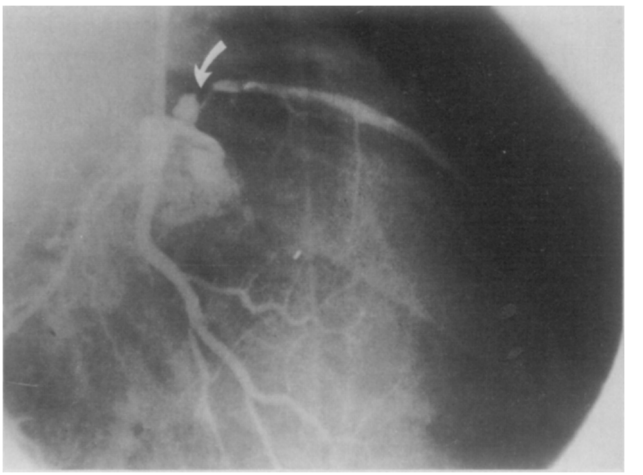

Fig. 3. Coronary arteriogram of the left anterior descending artery in the 30-degree right anterior oblique view. An abrupt and eccentric severe stenosis of the proximal artery with a normal distal vessel is seen.

occlusion following blunt chest trauma may be due to atherosclerosis,[3] spasm,[3] or embolic events.[9] In our patient, an intimal dissection was likely (Fig. 3), resulting in an obstructive arterial lesion. Repeat coronary arteriograms in several patients with coronary occlusion due to blunt chest trauma[3,5] have shown that the natural history of such lesions is similar to intimal injuries following cardiac catheterization,[10] frequently with complete healing of the lesion within 6 months. Therefore, in the absence of ongoing ischemia, medical therapy may be appropriate. Acute intervention with thrombolytic agents or coronary angioplasty has not been attempted in these patients,

perhaps because as in our patient, a long period of stabilization of concomitant injuries and the fear of bleeding precludes early catheterization.

In summary, we report an unusual case of coronary obstruction with subsequent myocardial infarction following blunt chest trauma. A definite diagnosis of myocardial infarction and an estimate of the extent of myocardial injury was provided by thallium perfusion scintigraphic techniques. Follow-up over 2 years has revealed stable symptomatology and ventricular performance despite cardiac chamber enlargement. To our knowledge, this is the first report of the use of thallium myocardial perfusion scintigraphy immediately following blunt chest trauma to ascertain the definite presence and extent of myocardial necrosis. As current techniques for the early and unequivocal diagnosis of cardiac injury and infarction following chest trauma are inadequate, further analysis of this technique is warranted.

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Preoperative localization of an intracardiac foreign body by two-dimensional echocardiography

Derek A. Fyfe, M.D., Ph.D.,*
James R. Edgerton, M.D.,** Amer Chaikhouni, M.D.,**
and Charles H. Kline, R.D.M.S.* *Charleston, S.C.*

From the *Department of Pediatric Cardiology and the **Department of Cardiothoracic Surgery, Medical University of South Carolina.

Reprint requests: Derek A. Fyfe, M.D., Ph.D., Dept. of Pediatric Cardiology, Medical University of South Carolina, 171 Ashley Ave., Charleston, SC 29425.

Accurate localization of intracardiac foreign bodies is essential for satisfactory operative removal. In some cases, despite localization by angiography, foreign bodies have been impossible to locate at operation.[1-3] Intracardiac foreign bodies may be accurately localized by two-dimensional echocardiography. By utilizing multiple echocardiographic windows, tomographic planes of the heart may be examined and the foreign body may be visualized in several planes. This case report describes the echocardiographic localization and successful surgical removal of an air rifle pellet buried in the ventricular septum.

A 13-year-old white girl was accidentally shot with a "pump" mechanism air rifle, the pellet passing through the left lateral chest into the heart. She remained hemodynamically stable. The entry wound was in the third intercostal space in the parasternal region. Chest x-ray examination localized the pellet in the center of the heart, both on anteroposterior and lateral views. Fluoroscopy showed the pellet to move with the heart during the cardiac cycle. Two-dimensional echocardiography was performed with an ATL (Advanced Technology Laboratories, Inc., Bellevue, Wash.) model 600 sector echocardiography system with a 5 MHz transducer. From the apical view (Fig. 1*A*), the pellet was visualized embedded in the interventricular septum adjacent to the tricuspid septal leaflet. From the left parasternal long-axis view (Fig. 1*B*), the circular foreign body was seen in the left ventricular outflow tract near the anterior mitral leaflet and just below the aortic anulus. From the left parasternal short-axis view (Fig. 1*C*), the pellet is seen in the ventricular septum of the left ventricular outflow tract subjacent to the right and noncoronary aortic cusps. The right and left atrial and ventricular cavities were interrogated with pulsed Doppler echocardiography and no evidence of mitral or tricuspid incompetence or of a traumatic ventricular septal defect was detectable.

At operation, examination of the heart revealed the entry wound medial to the left anterior descending coronary artery in its middle third. After opening the aorta and retracting the aortic valve, two fragments of fibrous debris were seen attached to the interventricular septum 5 mm below the right and noncoronary aortic cusps. These fragments were removed by sharp dissection. It appeared that these fragments were caused by the traversing pellet as it emerged from the left ventricular wall and went back into the interventricular septum. Following palpation of the left ventricle from inside the heart and palpating the interventricular septum from outside the heart, the pellet was detected exactly in the predicted position and was removed. The operation was concluded, and the patient made an uneventful recovery. Prior to discharge, two-dimensional and Doppler echocardiography showed no evidence of residual defects or fragments.

Retained intracardiac foreign bodies should be surgically removed to prevent subsequent endocarditis, pericarditis, embolization, fistula formation, or myocardial damage.[1-4] However, attempted removal is often unsuccessful due to failure to locate the foreign body.[1-3] This has occurred in approximately 30% of cases. Angiocardiogra-